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GENERAL ARTICLES

TUBERCULOUS LOBAR ATELECTASIS AND THORACOPLASTY

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On reviewing over 200 cases of complete thoracoplasties done at this sanatorium, a striking feature has been that the consistently good results have been obtained in a group of cases when the disease is of the consolidatory type, that is a state of segmental or lobar airlessness corresponding to the pre-fibrotic stage which has not yet reached complete fibrosis. It is important to establish a difference between these two degrees of lung fibrosis as they represent two consecutive phases in the process of healing, and the performance of a thoracoplasty in one or the other may have considerable bearing on the final results.

The role of atelectasis or consolidation as a healing factor in pulmonary tuberculosis has not been credited with the merit it deserves, and by some has even been considered as a complication. Despite literature and experimental work on the subject, many still associate atelectasis chiefly with bronchial obstruction due to organic material (blood clots, sputum plugs, bronchial secretions). This accident, which may render a whole lung functionless, together with the added danger of entrapped septic material within the blocked bronchi, is a well-recognised cause of pneumonitis and bronchiectasis. We readily agree that urgent relief of this obstruction with consequent re-aeration of the lung is the correct treatment of this type of atelectasis, but this temporary condition has little to do with the type of atelectasis that we are about to discuss.

In this latter type, atelectasis is really a misnomer, but the other terms suggested for this condition have in our opinion not improved upon it. This condition could hardly be called fibrosis, since this is a slowly developing process while atelectasis develops rapidly. It may appear that collapse or consolidation or pneumonitis are better terms, but again these imply rather a state of tissue consistency or hepatisation which does not exist here, since an atelectatic lobe is flabby, airless, dark in colour, light in weight and smaller in size. Therefore be the condition called atelectasis, detelectasis (Fleschner),

collapse, consolidation, interstitial fibrotic pneumonitis (Pinner), fibrosis, lobitis, etc., the fact is that it appears or can develop in a large number of cases of pulmonary tuberculosis, particularly during artificial pneumothorax treatment.

It would be quite easy to describe this pulmonary change as an autonomous clinical picture of pulmonary tuberculosis, and we are not sure that this has not already been done by some authors. Radiologically (and this is probably its most characteristic feature) it appears as a large, opaque area, usually occupying the whole of the upper lobe (the right more often than the left), with a well-defined lower border (the interlobar fissure) rather triangular in shape, with the vertex towards the root of the lung and the cavity or cavities and other tuberculous lesions imprisoned within. Tomography is the best way of assessing the number and size of those cavities, sometimes not very clearly visible in a plain X-ray owing to the density of the parenchymatous disease. Frequently there is some deviation of the trachea and mediastinal structures towards the affected lobe, and even the diaphragm can be noticeably raised. Serial X-rays show this opacity gradually contracting upwards, towards the upper mediastinum. With the shrinkage of the lobe, the cavities can be seen getting smaller and in some cases may even disappear altogether, particularly if a phrenic crush has been done, either alone or in conjunction with a pneumoperitoneum.

The general condition of these patients is usually good, and it is not uncommon for them to gain weight; they may have only very slight symptoms and physical signs are often few. A common important feature is the persistently positive sputum. Despite many papers on tracheo-bronchial tuberculosis, physical signs of bronchial stenosis were only clearly present in one of our cases which came up for thoracoplasty, and this case operated on two years ago is now one of our best results. Bronchoscopic examination has not been done as a routine in our cases, either before or after the operation, as it was found that bronchoscopy helped little in deciding whether or not surgical intervention was desirable. When bronchoscopy was performed it was found that the main pathological changes consisted of sub-mucous infiltration, œdema and hyperæmia of the mucus membrane. Bronchography, however, is another matter, and this will be referred to later on.

Returning to the atelectatic changes observed in some cases of lobar tuberculosis, Coryllos attributed this phenomenon solely to one factor—viz., the obstruction of the draining bronchi. If this obstruction is complete, the corresponding pulmonary segment or lobe becomes isolated from the atmospheric air and the air enclosed within this portion of the lung is gradually or rapidly absorbed by the blood stream. This brings about alveolar collapse and tissue anoxæmia, which in its turn makes the growth and life of the tubercle bacillus precarious or impossible. This bronchial obstruction, so essential for the production of atelectasis, has originated from the inflammatory condition of bronchial mucosa previously affected by tuberculous bronchitis. Although Coryllos' conception of cavity closure due to bronchial obstruction has been challenged by some authors, none can deny the widespread acceptance of his points of view, which today are almost universally recognised.

In lobar atelectasis the radiological opacity of the affected pulmonary

tissue can vary from a true homogeneous density (" black-out lobe "), in which the imprisoned cavity can be clearly seen, to a mere loss of normal translucency. In the first case a bronchogram would show bronchial obstruction at the level of the main divisional bronchi of the upper lobe; in the second it is possible that lipiodol may even reach the finest bronchi, demonstrating that bronchial obstruction is not the sole cause of atelectasis in these cases. However, both types of atelectasis can be beneficial in the healing of tuberculous lesions, provided they are skilfully used at the right time.

We believe that bronchial obstruction is an intermediate stage rather than an initial one in the development of atelectasis in pulmonary tuberculosis, and also that lobar fibrosis represents the end result of a long period of pulmonary airlessness.

Lobar atelectasis can be produced artificially in certain " predisposed " cases by means of artificial pneumothorax. These cases have lesions distributed in a fan-like manner, the interlobar fissure is clearly visible, and there may be two or three small cavities situated peripherally. This topographical distribution of the lesions has been termed the pre-atelectasis stage.

However, there are other means of producing lobar atelectasis. Adams, Van Allen, Brooks and others have been able to produce this condition experimentally, using mechanical and clinical agents, but when possible artificial pneumothorax is the simplest, and should it control the lesion, it may be all that is necessary. Should this happy state be attained, it is imperative that refills should be continued until the affected lobe has reached its maximum shrinkage, when the rest of the lung should then be allowed to re-expand and the corresponding hemi-diaphragm be paralysed and raised. To attain this a short period of pneumoperitoneum treatment may be necessary. If, however, artificial pneumothorax succeeds only in producing lobar atelectasis and the cavity or cavities remain open, the procedure should be abandoned in favour of a thoracoplasty. At this juncture we would like to make an observation on timing.

When an artificially collapsed lobe presents itself as an opaque mass containing uncontrolled cavities, is it advisable to abandon the A.P. immediately or should it be continued for a while?

There are several strong arguments against this latter policy. The danger of a pleural effusion is one, and this argument is further strengthened by the possibility of this effusion leading to an empyema or an unexpansible lung—a bronchopleural fistula being the common ethology of both these conditions.

However, most of our best results on cases of tuberculous atelectasis were found in the group where this atelectasis was the result of a period of pneumothorax treatment. And in these cases there is no doubt that the pneumothorax, even if complicated by a pleural effusion, and probably because of it, acted as a preliminary agent stimulating the proliferation of fibrous tissue and leading to the development of the so-called opaque hemithorax, where the whole lung appears shrunken (carnified lung).

This observation is corroborated by the work published by Sarno-Blanco on prognosis in atelectasis. According to these authors, if atelectasis was associated with a process of pachypleuritis or fibrous pleurisy, the prognosis was usually good. But if, on the other hand, the fibrosis was primarily paren-

chymatous, from exudative lesions, together with anatomical and functional changes of the bronchial tree, the evolution was unfavourable.

The position thus created by this succession of events, pneumothorax-atelectasis-pleural effusion, rather resembles the experimental atrophy and cirrhosis of the lung obtained by Wolfe-Wang and Van Allen on dogs, after enclosing lobes into loosely fitting envelopes of thin rubber which were then inflated. This caused intense pleural reaction and pleural thickening. As pointed out by Coryllos, persistent atelectasis on the one hand, and thickening of the visceral pleural on the other, leads to fibrosis because of persistent anoxæmia. It is worth noting that in these experiments hyperæmia and fibroplastic proliferation developed in the compressed parenchyma and that all small bronchial tubes and alveoli were replaced by scar tissue, leaving only the larger tubes intact, though these were collapsed. The shrinkage reduced the enclosed lobes to the size of a small, firm mass in a few months. The similarity of this result and the mechanism of its production to the development of pulmonary carnification is very striking. And it should also be added that in these experiments the mediastinum, hemidiaphragm and chest wall showed a tendency to obliterate the dead space left by the lobar shrinkage.

This extrinsic way of obtaining lobar atelectasis preparatory to fibrosis is, however, different from the way by which atelectasis and pleural effusion may follow artificial pneumothorax, but rather illustrates the importance of visceral pleural thickening as a factor of lung fibrosis.

In our series of 32 cases of tuberculous lobar atelectasis with cavitation submitted to thoracoplasty, the association of atelectasis and pleural effusion was clearly demonstrated in 12 cases, and the results are shown in Table I.

The importance of pleural thickening as a fibrosing factor in the healing of tuberculous lesions is clearly demonstrated in thoracoplasty, extrapleural pneumothorax and oleothorax. Of these, if our contention is correct, the one that establishes conditions under which atelectasis and pleural reaction are present together in a manner nearest to that of the intrapleural pneumothorax is extrapleural pneumothorax. Here, however, the dead space thus created would have to be obliterated by an early decostalisation in order to obtain a result similar to that of the perfect thoracoplasty, which still remains the best form of treatment for the condition that we are discussing.

It is an almost universally accepted principle in the treatment of pulmonary tuberculosis that the diseased lung should not be forcibly compressed if we can use the more relaxing methods provided by selective pneumothorax or extrafascial concentric apicolysis. To this we add our belief that the indication for surgical collapse therapy *par excellence* is when the lesions themselves show a tendency towards healing which for one reason or another cannot be completed—viz., rigid chest wall, negative pressure pull, condition of the bronchi. This is represented by tuberculous lobar atelectasis after the fissural hand of the hemithoracic clock has travelled from nine to ten o'clock and the mediastinal and diaphragmatic shutters have begun to close in.

Sauerbruck has said: " Lung tissue cannot be stimulated to resistance by active compression, and the active factors of healing should be left to the organism merely establishing conditions under which the lung can exert its spontaneous tendency for retraction. Before venturing on major surgery a

TABLE I

Cases	*Age*	*Sex*	*Lobes affected*	*Apicolysis*	*No. of stages*	*No. of ribs resected*	*Time since operation*	*Sputum after operation*	*General condition after opn.*	*Remarks*
1	20	F.	L. lung	Yes	3	1-8	5 yrs.	Negative	Good	Working
2	30	F.	L. lung	No	3	1-10	4 yrs.	Negative	Good	Working
3	22	M.	R. lung	No	3	1-10	4 yrs.	Negative	Good	Working
4	31	F.	L.U.L.	Yes	2	1-5	4 yrs.	Negative	Good	Working
5	20	F.	R.U.L.	Partial	2	1-6	3 yrs.	Positive	Poor	Bilateral thoracoplasty
6	32	F.	L. lung	No	3	1-11	3 yrs.	Negative	Fair	Housework
7	29	F.	L. lung	No	3	1-8	2 yrs.	Negative	Good	Housework
8	30	F.	L. lung	No	3	1-8	2 yrs.	Negative	Good	Housework
9	33	F.	R.U.L.	Yes	2	1-7	2 yrs.	Positive	Good	Housework
10	30	M.	R.U.L.	No	2	1-6	2 yrs.	Negative	Good	Housework
11	24	F.	L.U.L.	No	2	1-6	1 yr.	Negative	Fair	Housework
12	23	F.	L. lung	No	2	1-7	1 yr.	Negative	Fair	Housework

certain degree of demarcation of disease should have occurred and until then conservative treatment only is indicated."

Of the same opinion are most of the well-known authors. Thus Gravensen writes: " The chief field for collapse therapy is found in lung cases which show signs of healing by retraction owing to the production of fibrous tissue, pulling of the chest wall and displacement of the mediastinum, heart and diaphragm, when at the same time this process is prevented from being completed by the rigid condition of the chest wall."

This distinction between tuberculous lesions treated during a phase of regression, while natural healing mechanisms are still in action, and other lesions, where this process is complete, is a very important one, and this is borne out by any extensive follow-up of thoracoplasty cases. Thoracoplasty in itself does not produce healing of tuberculous lesions. As Abello Pascual has shown, " there is nothing specific in the parenchymatous changes that follow collapse therapy." According to Stefko, after a thoracoplasty, all that can be found is " detritus from the compressed lung, blood and lymph stasis, bronchitis, atrophy and necrosis of the small bronchi, development of connective tissue throughout the tubercles, pleural fibrosis and emphysematous changes of the non-collapsed pulmonary segments. The cavity may appear obliterated by the growth of granulating tissue slowly filling the dead space." There is nothing really specific in all these histological changes. In the neighbourhood of the cavity, destructive and reparative processes still continue. Dissemination of the disease may still occur even a long time after the operation.

Conversely, in most of the cases of lobar atelectasis treated by thoracoplasty once the diseased area had reached a given degree of demarcation and shrinkage, the follow-up of these cases shows that the disease was effectively controlled. However, some who were operated on four and five years ago can now be considered cured.

The statement of Dormer, Friedlander and Wiles that every thoracoplasty, even the successful ones, ends in residual bronchiectasis, and that this in itself is a sign of therapeutic failure, is too provocative to remain unchallenged. It is precisely this particular anatomical type we are discussing here—tuberculous lobar atelectasis—the one which some authors more particularly associate with bronchiectasis. The above-mentioned authors state that bronchiectasis is present before the operation, and that this persists after thoracoplasty. Two cases of thoracoplasty for this very condition we are discussing here are presented by them in their paper. The accompanying bronchograms leave no doubt as to the presence and persistence of bronchial dilatation within the blacked-out lobes. Yet sputa were rendered negative and the operations were classified as very successful.

Bronchiectasis following thoracoplasty, even bronchiectasis due to fibrotic lesions, unless of the suppurative type, is a benign phenomenon in the process of healing and can actually be a manifestation of the healing process itself. Bronchiectatic changes in atelectatic lobar tuberculosis, as shown by bronchography alone, do not, as a rule, affect the small bronchi as in non-tuberculous bronchiectasis. Rather the larger ones are affected, due primarily to lesions of all the layers of the main bronchial walls, and their presence after a thoracoplasty may be responsible for the raising of small amounts of sputum which

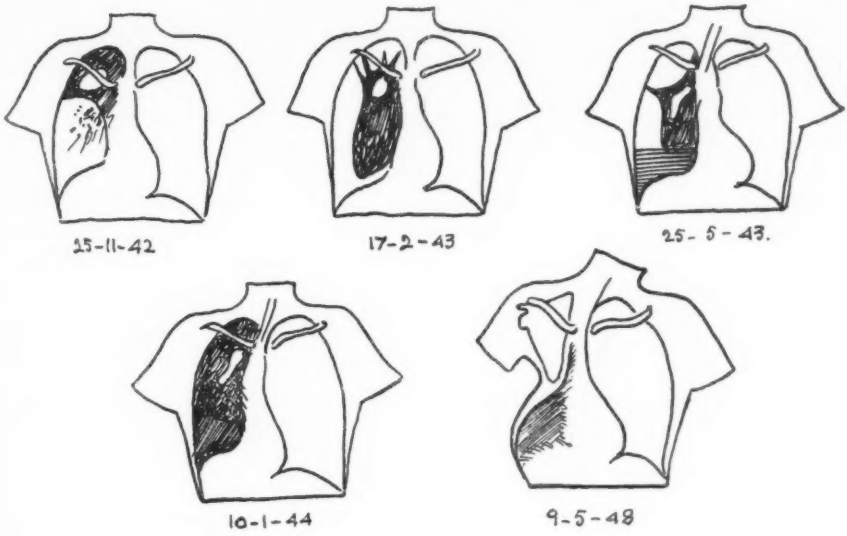

FIG. 1.

Brief clinical notes: Admitted on 19.11.42 with positive sputum and a radiological picture of right lobar tuberculous consolidation with a medium-size cavity and scattered infiltration on remaining lobes. Right artificial pneumothorax induced on 19.1.43. Right phrenic nerve crush on 24.4.43. Developed fluid and required repeated aspirations. Three-stage thoracoplasty in 1944. Since then quite well, working, with negative sputum and very slight symptoms.

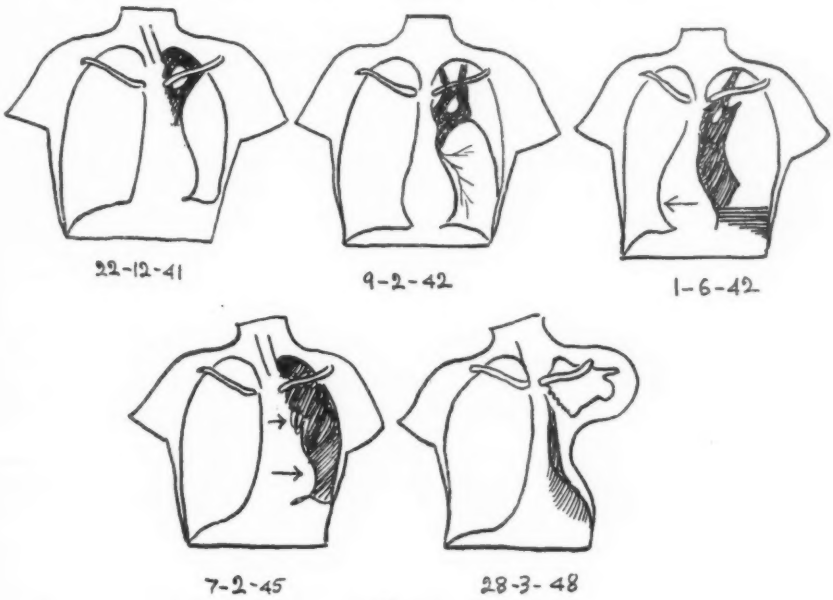

FIG. 2.

Brief clinical notes: Admitted on 5.2.41 with positive sputum and a radiological picture of left upper lobe tuberculous consolidation with cavitation. Left artificial pneumothorax induced on 22.1.42. Left phrenic nerve crush on 5.5.42. Developed fluid and required some aspirations. Had physical signs of bronchial stenosis. Three-stage thoracoplasty in 1946. Since then negative sputum.

is negative for the tubercle bacillus. It is the collapse of the small branches which is the important point in the isolation of the tuberculous foci.

The usefulness of thoracoplasty operations in these cases is that by removing the rigid framework of the costal arches the shrinkage of the consolidated lobe is increased, anoxæmia and fibrogenesis encouraged, and toxic absorption minimised.

M. Pinner has paid particular attention to the factor of pyogenic infection in atelectasis, and is also of the opinion that in the absence of purulent secondary infection the atelectatic lung tissue obtained by collapse therapy is a preliminary stage in the fibrotic encapsulation and healing of tuberculous lesions. In the case of tuberculous cavities surrounded by atelectatic tissue, P. M. Andrus has stated that " the atelectatic shrinkage of the pericavital disease is probably more important in obtaining cavity closure than the bronchial blockage factor defended by Coryllos, and that the chief benefit of collapse therapy consists in permitting atelectatic shrinkage of the diseased lung to proceed."

Conclusions

Lobar atelectasis, far from being an accidental or complicating occurrence in pulmonary tuberculosis, plays a very important role in the mechanisms of healing by fibrosis.

Of the three main types of lobar atelectasis which can be seen in the course of certain cases of pulmonary tuberculosis, acute or malignant, sub-acute or benign, and chronic, the latter is the most suitable for thoracoplasty treatment. It may appear as a gradual development from the other two types or as a consequence of previous pneumothorax treatment. When it follows the latter the succession of events in this order—pneumothorax-atelectasis-pleural exudate—has been found to have a favourable effect on the resolution of the pulmonary lesions provided that it has been supplemented by thoracoplasty. This seems to be due to the fibrogenic role of the pleura in the gradual development of pulmonary shrinkage. It is considered that atelectasis precedes and encourages fibrosis. Thoracoplasty, by further assisting the retractive processes shown by lobar atelectasis, establishes conditions by which the active factors of natural healing are completed.

Atelectasis is probably the most specific reaction in pulmonary tuberculosis, and is Nature's effort to limit the extension of the disease. Its occurrence should be encouraged by all means at our disposal. Once atelectasis has developed and shrinkage of the diseased lobe has begun, thoracoplasty should be kept ready at hand to be undertaken at the most suitable moment so that this favourable position can be converted into a permanent advantageous gain.

I wish to acknowledge my thanks to Dr. H. G. Trayer, Medical Superintendent, Baguley Sanatorium, and Mr. A. Graham Bryce, Consulting Thoracic Surgeon, for their extreme kindness and great facilities afforded; and to Dr. V. E. Sherburn, Deputy Medical Superintendent, High Carley Sanatorium, for his help in preparing this paper.

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MILIARY CARCINOMATOSIS OF THE LUNGS

REPORT OF A CASE SIMULATING MILIARY TUBERCULOSIS

BY JOHN LISTER

From the Royal Free Hospital

MILIARY carcinomatosis of the lungs was first described by Andral (1829) and later by Virchow (1855), but the first full descriptions of the condition were given by Troissier and Raynaud, both in 1874.

The condition is now well recognised and always taught to students as one of the classical causes of a snow-storm appearance in chest X-rays. There have, however, been only relatively few cases reported in the English literature, the majority coming from France, Germany and the United States.

In a review of the literature up to that time, Wu (1936) discussed the clinical and pathological features of 49 cases. Of these cases, 32 were in males, 18 in females and the age incidence varied from 22 to 77, 18 occurring in patients under 40. The stomach was the primary site in 36 of the cases, the bronchus being the second most common site, but accounting for only 5 of the cases. Other primary sites were the breast, prostate, uterus, sigmoid colon and gall bladder.

Pathology

There is apparently no relation between the histological type of the primary growth and the occurrence of miliary carcinomatosis of the lungs (Wu, 1936).

In his paper in 1874 Raynaud described the condition as a diffuse invasion of the lymphatics of the lungs with cancer cells, without the appearance of cancerous nodules of any size in the lung parenchyma. Troissier described the condition as pulmonary lymphangitis carcinomatosa. The primary emphasis is thus on the lymphatic nature of the spread, and it is commonly accepted that this occurs by retrograde lymphatic permeation caused by blockage of the lymph ducts in cancerous lymphatic glands. The high incidence of its occurrence in carcinoma of the stomach might suggest that this was due to the proximity of this organ to the thoracic duct, but the fact that carcinoma of the stomach is one of the commonest of all malignant new growths must not be overlooked. Moreover, spread through the thoracic duct would imply that the transmission of the tumour cells to the lungs was chiefly via the blood stream after they had been poured into the venous system from the thoracic duct.

The development of miliary nodules throughout the lung fields is due either to the escape of tumour cells from the distended lymphatics or to blood-stream spread, and it would seem reasonable to suppose that in cases of widespread dissemination, especially when other organs such as the liver and adrenal are affected, both the lymphatics and the blood stream are carrying tumour cells.

Von Meyenburg (1919) was the first observer to draw attention to the compression of the smaller bronchi by peribronchial and perivascular infiltration and the occurrence of endarteritis of the vessels in the vicinity of invaded lymphatics. These are the changes which are no doubt chiefly responsible for the pulmonary hypertension which develops in some of the cases, causing the chronic cor pulmonale with the intense dyspnœa and cyanosis described below.

Clinical Features

It is interesting that in cases of miliary carcinomatosis of the lungs the primary neoplasm is frequently silent and the most common presenting symptoms are cough, dyspnœa and cyanosis. The dyspnœa may be intense (Gaines, 1935), and this and the cyanosis are probably due to two factors: the first, pulmonary, due to diminution of aerating lung surface, and the second, cardiac, due to right-sided heart failure, a few cases even having presented as chronic cor pulmonale (Greenspan, 1934). That the conditions may simulate miliary tuberculosis was noted by Lambie and Collier (1946) in reporting a case, similar in many respects to the one reported here.

The patients are usually in an advanced state of cachexia, but it is a striking feature that clinical examination of the chest may yield little clue as to the extent of the pulmonary involvement. In the case described below there was a large effusion on the right, but there were no abnormal signs on the left. Likewise radiology may not always reveal the changes which, when fully developed, are so characteristic. In early cases the lung fields show only an increased reticulation, probably due to the distension of the lymphatics with tumour cells and to the peribronchial and perivascular fibrosis. Later a definite granular appearance is seen throughout the lung field, the granules increasing in size until the typical snow-storm appearance develops. Diagnosis is therefore frequently difficult, and it is a feature of many of the cases described that the diagnosis has only been made late in the disease or on the autopsy table. Perhaps one of the causes of the diagnosis being missed in the early stages is the frequency with which the primary growth is silent, and the fact that one is dealing with a malignant process may be overlooked altogether, as happened in the case reported below.

Case Report

Cpl. S., aged 25, of the Royal Tank Regiment, was admitted to a British military hospital in Germany on May 3, 1948, complaining that for the past month he had had a cough with a small amount of sputum, shortness of breath on exertion, excessive fatigue and insomnia. He had also had one small hæmoptysis, had been losing weight and had occasional pain across the shoulders.

The only relevant past history was an attack of pneumonia in Syria in 1943, but he did not know which side was affected and no details were available. A

brother had died of pulmonary tuberculosis in 1937, but apart from this the family history was negative.

At the time of his admission to the military hospital in Germany he was slightly febrile, temperature 99°, and had a tachycardia to 88. There was no finger clubbing and no cyanosis. Examination of the chest showed diminished movement at the right base with diminished vocal fremitus and vocal resonance, impaired percussion note and absent breath sounds over the same area. There were inspiratory rhonchi over the right upper lobe and mid-zone. Chest X-ray showed collapse of the right lower lobe. The sputum was negative for tubercle bacilli and he was treated with penicillin and breathing exercises, but the collapse of the right lower lobe persisted and further X-ray showed that the collapse had extended to involve the right upper lobe. Breathing exercises were therefore discontinued, and when on May 20, 1948, it was thought that a right pleural effusion was developing, diagnostic aspiration was performed and 30 c.c. of pale straw-coloured fluid were removed through the second right space posteriorly.

Examination of this fluid was as follows:

"Sp. gravity 1020; cells 17,000 per c.mm., 74 per cent. being lymphocytes; no organisms seen; culture sterile."

At the end of May 1948 he was evacuated to the United Kingdom and was admitted to a military hospital with the diagnosis of collapse of the upper and lower lobes of the right lung and a right pleural effusion.

On June 5, 1948, 800 c.c. of clear straw-coloured fluid were aspirated from the right chest and it was noted that many large endothelial cells were present, but again no organisms were found and culture remained sterile.

On June 8 he was transferred to another military hospital where the physical signs were those of a large right-sided pleural effusion, but it was noted that the mediastinum was displaced to the right in spite of the presence of the fluid. Chest X-ray there showed miliary shadowing throughout the left lung and it was considered that he was suffering from miliary tuberculosis, and he was transferred to the Royal Free Hospital on June 16, 1948, for streptomycin treatment.

On admission he was febrile (temperature 99°), had a tachycardia (pulse 100) and the respiration rate was raised to 30. He looked toxic and wasted. Physical signs in the chest were again of a massive right-sided effusion, the percussion note being stony dull and the breath sounds being absent on the right except at the apex, where they were bronchial in character. There were no abnormal signs in the left lung and no enlarged axillary or cervical lymph glands. The sputum was negative for tubercle bacilli, the E.S.R. was 13 mm. in the first hour, the hæmoglobin 83 per cent., and chest X-ray confirmed the presence of a massive effusion on the right and showed the miliary shadowing throughout the left lung seen in Fig. 1.

A small amount of pleural fluid was removed for further examination and this was again straw-coloured, contained a large number of cells, 83 per cent. of which were lymphocytes and 17 per cent. large endothelial cells. No tubercle bacilli were seen and culture, including a Loewenstein inoculum, remained sterile.

In view of the history and clinical picture, it was considered that, in spite of the failure to isolate tubercle bacilli, the diagnosis was most probably miliary tuberculosis, and on June 19, 1948, a course of streptomycin 1 gm. intramuscularly b.d. was initiated.

No improvement took place in the clinical condition, but, on the contrary,

progressive deterioration occurred. Nausea and anorexia were prominent symptoms, but thought to be chiefly due to the streptomycin. The patient became gradually weaker and in August 1948 complained of severe pain in the back. X-ray of the spine showed collapse of the 9th thoracic and 3rd lumbar vertebræ. This was considered to be most probably due to tuberculous disease of these vertebræ. Lumbar puncture was performed, but the fluid was found to be entirely normal.

The course of streptomycin was continued until September 25, 1948, when a total of 200 gm. had been given. After completion of the course, the condition continued to deteriorate, the patient became extremely wasted, exhausted and dyspnœic; chest aspiration was performed on two occasions owing to the increasing dyspnœa and slight symptomatic relief obtained, but he died on October 5, 1948.

Necropsy Report

The body was that of an emaciated man of 25 years. Apart from slight superficial ulceration of the face, there were no noteworthy external marks. There was a straw-coloured effusion of 2 pints in the right pleural cavity, and the right pleura was adherent to the chest wall. There was an effusion of about 1 pint in the left pleural cavity. The right lung was collapsed and felt solid; on section it was found to consist of a large number of discrete white firm nodules of about 1 cm. in diameter (Fig. 2). These bulged slightly when cut. Between them was consolidated lung tissue. None of the nodules was softened, and none was found to ulcerate into a bronchus. There was moderate enlargement of the right hilar glands. The left lung was not collapsed; it had much the same structure as the right lung, with numerous firm nodules of growth interspersed with healthy-looking lung tissue. No ulceration of the bronchi was discovered. There was a straw-coloured pericardial effusion of a few ounces. The heart was normal. There were a few spherical white masses in the liver, up to 1 cm. in diameter. Similar nodules were present in the cortex of both suprarenals. No other organ showed secondary deposits, and no other organ showed any noteworthy abnormality.

Histology

The nodules in the right lung consisted of a pleomorphic, papillary and acinar mucous secreting carcinoma, certainly bronchogenic in origin (Fig. 3). The adjacent lung tissue was infarcted. The nodules of the left lung and the deposits in the adrenals were of similar structure. Those in the liver were more anaplastic. There was a miliary carcinomatous infiltration of the epicardium, and there was infiltration into the marrow of a lumbar vertebra. "Heart failure" change was present in the left lung and there was some congestion and hæmosiderosis of the spleen. None of the other organs examined showed any secondary infiltration.

Discussion

This case is reported as one of misdiagnosis, and the purpose of the report is to see whether, in spite of the striking resemblance of the case to miliary tuberculosis, the correct diagnosis of miliary carcinomatosis could have been made.

When the patient was admitted to the Royal Free Hospital the radiological

PLATE IV.

FIG. 1.

X-RAY APPEARANCES IN JUNE, 1948, SHOWING MASSIVE RIGHT-SIDED EFFUSION AND MILIARY SHADOWING THROUGHOUT LEFT LUNG.

To face p. 34

PLATE V.

FIG. 2.

MACROSCOPIC APPEARANCE OF RIGHT LUNG, SHOWING MULTIPLE SMALL NODULES OF GROWTH.

FIG. 3.

SECTION SHOWING THE PLEOMORPHIC PAPILLARY AND ACINAR MUCOUS SECRETING CARCINOMA.

appearances were those of Fig. 1, the earlier radiograms showing the appearances of right lower lobe and later right upper lobe collapse only coming to light later, after the diagnosis of miliary tuberculosis with which he was admitted had been accepted and streptomycin treatment initiated. The evidence for the diagnosis of tubercle was admittedly slender, but the age and general condition of the patient, the presence of a pleural effusion and the appearance of miliary shadowing throughout the left lung seemed to suggest that it was the most likely diagnosis in spite of the failure to isolate the tubercle bacillus.

It is clear, however, that the presence of collapse of the right lung should have made bronchoscopy the first step in the establishment of a diagnosis and it is unfortunate that this was at no stage carried out. Nevertheless, even this measure would probably have failed to establish the diagnosis, as autopsy showed that there was no macroscopic ulceration of any of the main bronchi. It would therefore seem that to have made a firm diagnosis was virtually impossible, and it would have been more tragic to have failed to treat a case of miliary tuberculosis with streptomycin than to have mistreated a case of malignant disease, which in a patient of this age and of this extent must have been beyond any hope of recovery.

In retrospect it is perhaps right to say that the failure to isolate the tubercle bacillus should have cast more doubt on the presumptive diagnosis, and, secondly, the importance of complete reassessment of any case taken over from another hospital by transfer should be emphasised. Apart from these two points it does not seem possible to make further comment.

Summary

1. The subject of miliary carcinomatosis of the lungs is briefly reviewed with notes on the pathology and clinical features of the cases reported in the literature.

2. It is stressed that diagnosis is frequently difficult and often made only late or on the autopsy table.

3. A case is described in a male patient aged 25 years which so closely simulated miliary tuberculosis that it was diagnosed as such and treated with streptomycin.

4. The site of the primary growth appeared to be the right lung.

5. It is stressed that the signs and X-ray appearance of collapse of the lung should make bronchoscopy an essential step in the establishment of a diagnosis, but in this particular case the investigation would probably have failed to reveal any abnormality.

6. It is suggested that miliary carcinomatosis of the lungs may so closely resemble miliary tuberculosis that differentiation on clinical grounds can be virtually impossible.

I wish to thank Dr. F. P. Lee Lander for permission to publish this case, Dr. H. I. Winner for the morbid anatomy and histology and Mr. J. S. Sheppard for the photographic plates.

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MASS RADIOGRAPHY AND AFTER*

A PLEA FOR A MOBILE CHEST SERVICE

BY E. G. W. HOFFSTAEDT

From Holywood Hall Sanatorium, Wolsingham.

WITH an increasing number of Mass Radiography Units at work throughout the country the chances of developing an efficient case-finding programme have greatly improved. New recommendations for the future use of mass radiography (N.A.P.T., 1949) have taken into account accumulated experience and fresh views derived from this (Brailsford, 1945; England, 1946; Heaf, 1946; Hoffstaedt, 1944, 1947; Hoyle, 1945; Stradling, 1948; Toussaint and Pritchard, 1943; Trail, 1942).

Already an ever-increasing number of cases of tuberculous and non-tuberculous chest diseases are coming to light and others only await the application of the method on a still wider scale. Improved diagnostic facilities, however, are worse than useless if new cases cannot be given proper facilities for treatment. Waiting lists, already dangerously long (Bentley, 1948), are going to become still larger in view of the failure to find accommodation for far too many candidates. According to a recent statement by the Minister of Health in the House of Commons (April 28, 1949), no less than 11,700 tubercular patients are waiting for sanatorium admission, while at the same time 5,670 sanatorium beds are out of commission. As general hospitals, also desperately short of staff, have at present 21,600 beds closed, they will hardly have any beds to spare for our patients. With ever more non-tuberculous chest and heart diseases being discovered by mass radiography, the pressure on beds, both in general hospitals and in thoracic surgery centres, is bound to increase correspondingly. The limited bed accommodation for major chest surgery has to be shared between tuberculous and other chest and heart cases. In order to relieve the high pressure on the thoracic surgery centres the tendency is now to install more chest surgery units within the precincts of suitably equipped

* Paper read before the North of England Tuberculosis Society at their Annual Meeting, April 30, 1949.

and staffed sanatoria. This arrangement, however, results in a reduction of a corresponding number of beds, otherwise available for less advanced cases. Yet it is precisely this very type of case we are out to discover by mass radiography ! So, with the improvement of our diagnostic and therapeutic methods, we have actually created a vicious circle. How can we break it in order to find more beds, particularly for the " early cases "?

Brooke and Wetenhall (1949) came to the conclusion that the " facilities of out-patient departments have not been sufficiently developed as one of several means of affording some relief on bed pressure." On the other hand, many of our dispensaries and " clinics " fall far short of the minimal requirements. These mere " visiting stations " (Ridehalgh and Aspin, 1949) cannot fulfil the function of a chest clinic. The position is more favourable in great cities, where some enterprising chest physicians (Pritchard, 1948; Toussaint, 1948; Maclean and Gemmill, 1948) opened up a new avenue of approach. As a way out of the dilemma they offered the compromise solution of carrying out a form of domiciliary collapse treatment, partly in the patient's home, partly in well-equipped out-patient departments under X-ray control. It cannot sufficiently be emphasised that pneumothorax treatment without regular X-ray control is a very dangerous gamble and should not be recommended even as an expedient.

Though it is planned to move ill-equipped dispensaries and chest clinics, wherever possible, into out-patient departments of conveniently placed general hospitals, this most desirable policy is not easily put into practice. Moreover, it is practically impossible to equip adequately within reasonable time even half of those obsolete " visiting stations." However, the control of tuberculosis has become such a mass problem that it has to be approached by methods particularly suited to so big a task. It has been recognised that the way to cope with mass problems of this kind is the " mobilisation " of those scarce commodities, personnel and equipment. It will therefore become necessary—at least for some time to come—to operate modern chest clinics, adequately equipped for diagnosis as well as for treatment of pulmonary tuberculosis, and staffed by highly skilled personnel, as *Mobile Units*. Mobile Mass Radiography Units, Mobile Dental Clinics, Mobile Surgeries,* Mobile Physiotherapy Units and different kinds of specialists' " flying squads " form already a feature of the modern Health Service. The Mobile Chest Clinic, installed in a caravan or in a specially built coach, is, I venture to suggest, a practical solution for such districts—mostly rural and semi-rural areas—where well-equipped dispensaries or suitable out-patient departments of a modern hospital are not readily available.

The following is an outline of a working scheme suggested for the operation of a Mobile Chest Service.

Mobile Chest Clinic

A Mobile Chest Clinic (M.Ch.Cl.) (Figs. 1, 2 and 3) contains in one compact motorised unit an X-ray screening set in a dark room, also suited for laryngoscopy, etc., and a complete theatre for collapse treatment, chest aspirations and for all the usual clinical examinations (blood tests, gastric

* *Lancet* (1948), **2**, 749.

lavage, vital capacity estimation, etc.). The X-ray screening set can be operated from any standard 15-amp. power plug on the premises of any existing stationary " clinic " by means of a sufficiently long connecting cable fitted to the rear of the vehicle. The current from the mains might also feed electric radiators and fans for ventilation and will supply the electricity for lighting purposes, provision being made for emergency lights from the car battery. The theatre is fitted with a wash basin, a gas-operated water heater and a gas-operated instrument steriliser, the gas being supplied from Calor gas cylinders. A 50-gal. water tank, operating table and instrument tables and cupboards, oxygen outfit and fire extinguisher, can easily be accommodated in the generously lit theatre compartment. The dark room is entered through an electrically controlled light trap. Dressing cubicles are situated between the two compartments, each of which has an emergency exit.

The M.Ch.Cl. is specially designed for operating in conjunction with any existing, but poorly equipped, " dispensary " (or " clinic ") by being parked beside, or in front of, the dispensary with which it is connected by cable. The stationary dispensary functions as hitherto—*i.e.*, as waiting room and office, or as " visiting station " with the Health Visitor or Dispensary Nurse taking histories and keeping records and dealing with the patients in the usual way. The personnel of the M.Ch.Cl. consists of the Physician in Charge and/or his Assistant Physician, a T.A. Theatre Nurse (preferably S.R.N.) and an orderly who assists during the session—*e.g.*, marshalling the patients. It is, of course, essential for the proper functioning of the M.Ch.Cl. that an ambulance and a shooting brake should be attached to the unit for carrying ill patients to and from the clinic. The M.Ch.Cl. has two main functions to fulfil:

(i) To convert quickly, easily and most economically any existing strategically placed but unequipped " clinic " (dispensary) from a mere " visiting station " into an up-to-date chest clinic suited for both diagnosis and treatment of chest diseases. In spite of the lack of facilities for radiography (the latter would involve a much more ambitious and expensive scheme !) screening alone serves two essential purposes:

(*a*) To sift the radiologically negative from the positive cases. Hereby a great deal of superfluous X-ray films would be saved and so would a lot of unnecessary travelling for the patients.

(*b*) To establish some sort of preliminary diagnosis on the spot. The patient can be told at once whether or not there is anything seriously wrong with his chest—from the radiological piont of view—and much anxiety can be relieved at once. The physician will be able in most cases to give the patient a better idea of the situation at a first visit, and of fitness for work, need for further examinations and whether sanatorium treatment is likely to be necessary.

(ii) The M.Ch.Cl., as designed, would give much wider scope for clinical work than exists at present in so many instances. It also would allow those in charge to do most of their own diagnostic work as well as treatment. This opportunity opens up at once two significant aspects:

(*a*) Patients under collapse treatment will not have to travel, as so often

PLATE VI.

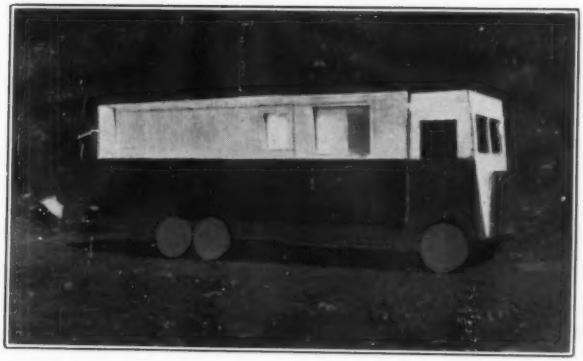

FIG. 1.
MODEL OF MOBILE CHEST CLINIC.

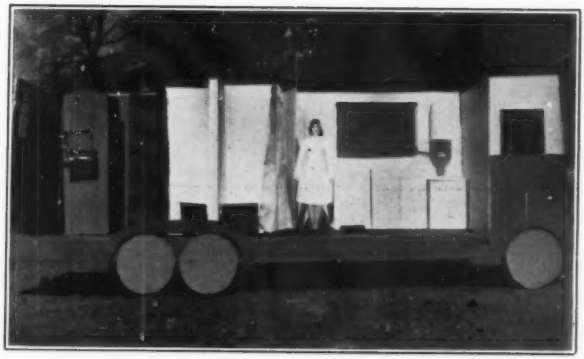

FIG. 2.
MODEL OF MOBILE CHEST CLINIC WITH TOP AND FRONT REMOVED.

To face p. 38

PLATE VII.

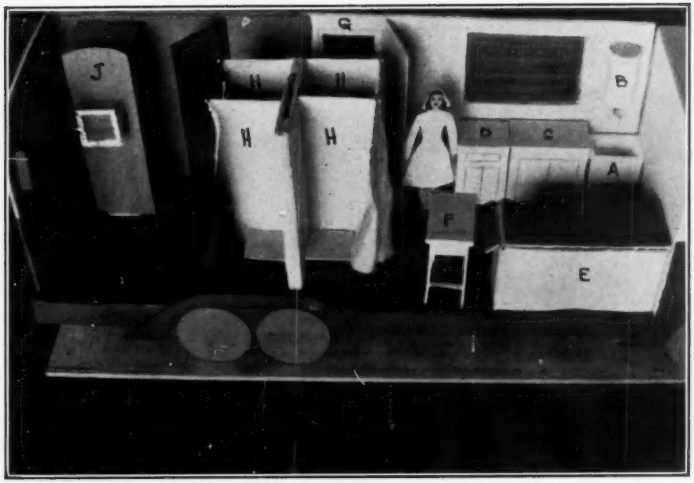

FIG. 3.

INTERIOR OF MOBILE CHEST CLINIC.

A.—Washbasin. B.—Hot Water Cistern. C.—Instrument Table and Cupboard.
D.—Desk. E.—Examination Couch and Cupboards. F.—Instrument Trolley.
H.—Cubicles. J.—Screening Unit.

they must, to distant treatment centres. The new arrangement will, apart from saving fares, save the patient fatigue, discomfort and risk.

(*b*) Even more important is the fact that with the opportunity of carrying out collapse treatment under X-ray control at the nearest " strategically placed dispensaries " a new enterprising scheme of combined " domiciliary dispensary " treatment on a large scale would become available. It might well allow mostly " early cases " to avoid long-stay sanatorium treatment. Such a scheme, as already in operation in a few progressive chest clinics (London, Leeds, Glasgow), has proved its value in relieving appreciably the increasing pressure on sanatorium beds. If such a scheme, so far the privilege of a few well-equipped clinics in great cities, could by means of M.Ch.Cls. gradually be extended over the whole country, sanatoria could put aside an appropriate percentage of their accommodation—say anything between 25 and 40 per cent. of their beds—for rapid admission and treatment of newly discovered " early cases." The improved diagnostic and follow-up facilities would keep most " observation cases " under dispensary observation and the conditions of new patients could be properly assessed before sanatorium admission is decided upon. Such a practice would in future free a significant number of sanatorium beds which at present are occupied by " observation cases." After a properly assessed case had been admitted to a sanatorium, collapse treatment, if decided on, could be started almost immediately and bronchoscopy or other clinical investigations could very soon be carried out. Division of adhesions or phrenic crushes being completed, A.P. patients could often within four to six weeks, P.P. patients probably within a fortnight, be returned home, *if* home conditions and the patient's general and chest condition permitted such a course. The economy in sanatorium beds which could be achieved by the suggested scheme would soon relieve pressure on sanatorium beds and would consequently reduce long waiting lists.

As to the routine work of the M.Ch.Cl. in regard to radiography it appears that Mass Miniature Radiography Units, if really mobile and if intelligently and economically operated, could take over a fair proportion of the clinic's radiographic work. This would incidentally provide adequate and comparable chest films ! The rest of the patients will have to be sent to the nearest X-ray centre as before.

Summary

Mass radiography and the increasing efficiency of our case finding programme, together with a growing demand for major chest surgery, are bound to aggravate an already dangerous situation. With 11,700 tubercular patients on the waiting lists and with 5,670 sanatorium beds out of commission, unorthodox methods of dealing with this mass problem need to be used. Encouraging results have been reported from various schemes of domiciliary and out-patient collapse treatment at well-equipped and suitably staffed chest clinics in big towns. Present unsatisfactory conditions make it impossible for many provincial dispensaries to adopt the same line of treatment, nor do they offer adequate diagnostic facilities. A modern Mobile Chest Clinic equipped with an X-ray screening set and a small theatre for refills, aspirations, blood tests, etc., is suggested as a compromise solution. It is pointed out how such

an arrangement would considerably relieve the pressure on sanatoria and reduce long waiting lists.

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My thanks are due to the following patients of Holywood Hall Sanatorium: to Mr. G. Bowman for drawing the original plans of the Model of a Mobile Chest Clinic; to Mr. R. Walmsley for building the Model according to my design; and to Mr. G. W. Henery for taking the photographs and producing the enlargements. Details of cost and delivery of the Mobile Chest Clinic (caravan type) can be obtained from Messrs. Hill Bros. (Hull) Ltd., 27, Park Street, Hull.

TRAUMATIC RUPTURE OF THE LEFT MAIN BRONCHUS

BY B. W. ANDERSON

IN an article describing two cases of traumatic rupture of the bronchus, Kinsella and Johnsrud (1947) reviewed the literature and gave details of 37 cases. Although this review may not have included all the known cases, the fact that they were able to find only 37 on record points to the rarity of the condition. Recognition of the condition at the time of injury occurs even more rarely and, including one of the authors' cases, only 4 cases are reported. Rupture of the bronchus is invariably caused by a severe chest injury of a crushing or compression type: many structures may be involved and the immediate fatality rate is high. Of the reported cases, 19 died either immediately or within a few days, and in nearly all, the ruptured bronchus was found post mortem. In 10 instances spontaneous recovery took place without any specific treatment and without the true nature of the injury being suspected at the time of the accident: in these a diagnosis was made at varying periods from 4 months to over 30 years later. Recently Fawcett (1949) described a case of rupture of the left lower lobe bronchus, discovered some 18 months after the injury, and successfully treated by lobectomy.

The object of this communication is to report a case in which spontaneous recovery took place with very little residual disability.

PLATE VIII.

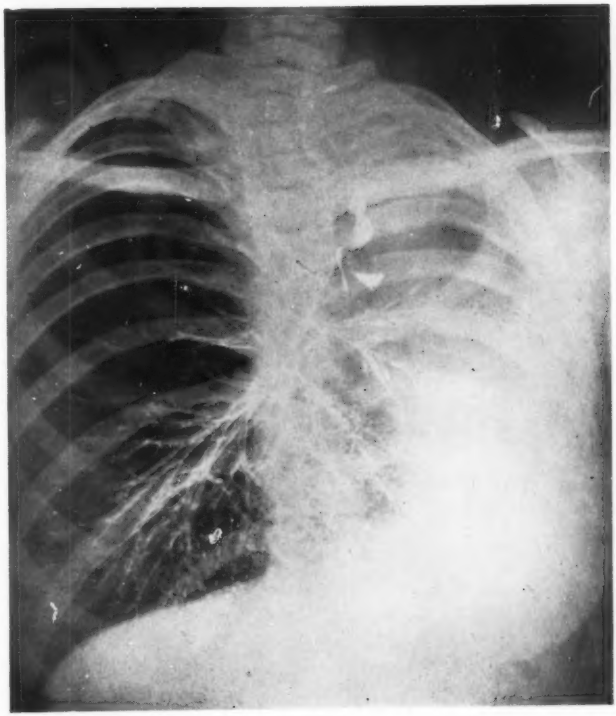

FIG. 1.

To face p. 40

In March, 1948, a girl, E.C., aged 16 years and 11 months, was sent to me for investigation. She gave a history of having contracted a cold three months earlier, and this had been followed by an irritating cough with some purulent sputum. Apart from the cough and a small amount of sputum she felt perfectly well and led an active life, being employed as a domestic servant and spending a good deal of her leisure time in dancing. She admitted having slight dyspnœa on fairly vigorous exertion. Her previous health had been good, but at the age of 9 years and 8 months she had been knocked down by a car and severely injured. Whether a wheel of the car actually passed over the chest is not clear, but there is no doubt there was a severe crush injury of the left chest. The accident occurred during the blackout on the evening of 3.12.40, and she was admitted to the Wemyss Memorial Hospital. She was dyspnœic, had extensive surgical emphysema and a fracture of the left clavicle, but no fractured ribs: a left hæmothorax developed which was aspirated twice. In spite of her critical condition on admission, she made a good recovery and was discharged from the hospital on 8.1.41. From that time until the onset of her present symptoms she had been well.

She was a tall, well-developed girl for her age and in good general condition. Inspection of the chest, on quiet respiration, revealed very little deformity, but on deep inspiration the shrinkage and fixation of the left hemithorax was marked. The percussion note was dull throughout the left side, the heart was displaced to the left, and, while distant bronchial breath sounds were audible in the upper zone, breath sounds were absent over the lower half of the lung. No adventitious sounds were heard and the right lung clinically was healthy. Routine examination of the sputum for tubercle bacilli was negative and the B.S.R. was normal.

X-ray showed a fairly uniform opacity over the lateral three-fourths of the left lung; the trachea was deviated to the left and the heart grossly displaced to the left and lost in the uniform opacity. There was no apparent disease in the right lung. Films taken after an attempt to introduce lipiodol into the left lung showed that the left main bronchus was completely obstructed 1 inch below the carina (Fig. 1): lipiodol had spilled over and outlined the lower lobe bronchi in the right lung, incidentally revealing the extent of hypertrophy of this lung. Bronchial branches from the right lung extended well into the left hemithorax.

The accident in December 1940 had caused a rupture of the left main bronchus with subsequent complete stenosis of the main bronchus and atelectasis of the lung. The case is of interest as one of spontaneous recovery with very little apparent disability.

I am indebted to Dr. John Wilson, Wemyss Memorial Hospital, for confirmation of her condition at the time of injury.

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REVIEWS OF BOOKS

Skeletal Tuberculosis. By VICENTE SANCHIS-OLMOS, M.D. Baillière, Tindall and Cox, London. Price 27s. 6d. net.

This survey is based on the author's experience in his native Spain, and upon a study of several hundred cases at the New England Peabody Home for crippled children.

The author's purpose has been to trace a pathological evolution for skeletal tuberculosis in harmony with that of the disease in the lung. To this end he has drawn from several sources not previously available in an English translation, notably that of J. Gonzalea Aguilar (Buenos Aires, 1940).

He accepts Ranke's conception of primary visceral infection followed by blood stream dissemination in the stage of secondary allergy, and also Redeker's " bud " theory, which goes far to explain the recurrence of local or distant activity so often seen after apparent cure of a skeletal lesion. To these he adds the period of " local hyperergy " to explain local extension of disease without general manifestations.

Three main pathological forms are recognised, the exudative, the proliferative (or granular), and the caseous. The distinguishing radiological features of these are, respectively, regional osteoporosis, localised rarefaction, and localised relative sclerosis.

The author's plan of treatment follows that of accepted custom. In general treatment, he regards as contra-indications to heliotherapy a fever over 100° F., an active pulmonary lesion and a sedimentation rate over 30 mm./hour. He warns against operative treatment in the secondary phase with local hyperergy, with the exception of extra-articular procedures such as Brittain's arthrodesis. The potential dangers of fibrous ankylosis are sufficient to demand arthrodesis in the inactive stage.

In discussing the juxta-articular focus, the author advises a conservative attitude, because of the dangers of operative treatment and because he is " somewhat resigned to the fact that little can be done to affect the evolution of a juxta-articular focus in the hyperergic phase."

This book provides some stimulating views on the pathological evolution of bone and joint tuberculosis, and a review of a part of the literature not readily available to the English reader.

Introduction to Diseases of the Chest. By JAMES MAXWELL. Third Edition. Hodder and Stoughton, London. Price 12s. 6d.

This well-known textbook, now in its third edition, has undergone little change since its first appearance in 1938. A section here and there reflects the advances of the last ten years; for the rest the author's teaching remains orthodox and is not likely to need short-term revision.

As an introduction to a special branch of medicine written for junior students, the book falls short of its aim. This is to some extent unavoidable, for the bewildering period of change from the lecture room to the wards is not the right setting in which to centre attention on any one system of the body. Continuity of training at this stage is of first importance and is best preserved by building on the twin foundations of Anatomy and Physiology. Diseases of the respiratory tract lend themselves less readily to this approach than many other topics in medicine; nevertheless, subjects like bronchial embolism, postural

drainage, dyspnœa, and pulmonary œdema could provide a useful link. Unfortunately, this book does not take advantage of these opportunities. It does, however, supply reliable information in a clear-cut form and dogmatic manner, suitable for examinations, and is likely, therefore, to enjoy continued popularity.

The opening chapters give an outline of the major symptoms, physical signs, and special investigations, while the remaining two thirds of the book are devoted to an account of the commoner diseases of the respiratory tract. The text is set in a rigidly classified form and includes several lists of the " diagnostic sieve " type. Although such lists can be misleading when not checked by clinical experience, in this instance the common diseases are given due prominence.

One of the best chapters deals with abnormal radiological appearances, which are rightly accorded equal status with physical signs. This aspect of thoracic diseases is perhaps more suitable than any other for description and illustration in a textbook, and the emphasis placed on it at an early stage of the student's career is welcome. The value of this section would be even greater if it included more detail on segmental lesions and on the features of the lateral view. The specimen radiograms are now printed as negatives and appear in larger format than in the first edition. In spite of this, the reproduction has suffered from the quality of the post-war paper. This has presumably influenced the author in his choice of the illustrations, many of which represent gross lesions. Others which could be replaced by better examples are: the normal bronchogram, with inadequate filling of the bronchial tree, the X-ray of a thoracoplasty marred by gross scoliosis, and the two pictures of artificial pneumothorax, neither of which shows really satisfactory collapse.

One might hesitate to recommend this book to students entering the wards for the first time. On the other hand, those who have already acquired a thorough grounding in general medicine will find it useful reading for the final examination.

P. F.

Tuberculosis in Childhood. By DOROTHY S. PRICE. Second Edition. John Wright and Sons, Ltd., Bristol. Price 25s.

It is a pleasure to read this lucid and well-constructed monograph. In it Dr. Price presents a comprehensive picture of tuberculosis in childhood and adolescence, based on her own experience and on an extensive study of the literature.

The main body of the work is devoted to a description of pulmonary tuberculosis and its sequelæ, divided along the usual lines into three groups: the primary complex, blood-borne infection, and bronchogenic lesions. Extrapulmonary tuberculosis is dealt with in a section written in co-operation with Mr. H. F. MacAuley, who also contributed the final chapter on tuberculous orthopædic lesions. An excellent survey of the epidemiology, pathogenesis, and diagnostic methods introduces the clinical section of the book, which is well documented with statistics, illustrative case histories, and a full bibliography.

Few would disagree with the author's conception of the pathogenesis and clinical picture of tuberculosis in childhood. Her views on the highly controversial subject of the development of pulmonary tuberculosis in the young adult will command less general approval. Dr. Price shows that in many western communities, as a result of better care, about 50 per cent. of adolescents

have still to face their primary infection at the age of 14 years. She believes that a considerable proportion of the casualties from phthisis in the 15-25 age group are a direct result of a late-acquired primary infection, while a very few cases follow reawakening of an incompletely healed hæmic focus dating back to earlier life. Exogenous superinfection is, in her opinion, exceptional.

The practical consequences of this conception of the disease, if it could be proven true, would be far reaching, for its logical sequel is that adequate healing of the primary complex and B.C.G. vaccination could provide us with the key to the control of phthisis. One would wish that the solution of this problem was really so near. Unfortunately, the evidence in favour of this theory is scanty and there is obviously much need here for further research.

Apart from this issue, all other criticism of the book concerns minor points. A work of this quality deserves production in larger format; the print is at present too small for comfortable reading and the illustrations of X-rays are not clear. A better diagram of the lymphatic system of the lung could also be substituted. As regards the text, more stress might be laid on the possibility of bronchiectasis following collapse and more detail given about the use of streptomycin in miliary tuberculosis and tuberculous meningitis. Nevertheless, the book is a storehouse of reliable information, and will be of value to all who are interested in the preventive and clinical aspects of tuberculosis. P. F.

O detske Plicni Tuberkulose (*Tuberculosis of the Lungs in Childhood*). By VLADIMIR VOJTEK. OLOUMOIC C F.P.S. Pp. 545. Price not stated.

Dr. Vladimir Vojtek, Medical Director of one of the largest children's sanatoria in Czechoslovakia, gives a very full account of all forms of tuberculosis affecting the lungs in childhood. The book is, for Central European conditions, extremely well produced, and some 214 excellent X-ray reproductions, each accompanied by a diagram, provide the illustrations for each of the five parts. The first of these deals exhaustively with diagnostic methods, clinical, radiological, physiological and bacteriological. The second part gives an account of all pulmonary manifestations of primary infection. The third part deals with what we would call the early post-primary period. The fourth part, perhaps the most important, deals with cavitating or destructive processes as they occur at the various stages of tuberculosis in childhood from the cavitating primary to the adult type of the disease. The fifth and last part deals with differential diagnosis and classification of primary tuberculosis in childhood, quoting in the original language the classifications used by the Anglo-Saxon, French and German schools.

The exposition is clear and concise. The views expressed are based to a very great extent on personal experience and show clearly the influence of pre-war German and French and post-war Anglo-Saxon and Scandinavian literature. Much importance is attached to the rôle of superinfection as the cause of malignant primary lesions. The evil influence of repeated or continuous massive exposure is particularly stressed. The book is the first comprehensive work on the subject in Czech. E. N.

Brompton Hospital Reports. Vol. XVI, 1947. Gale and Polden, Aldershot. Price 10s.

Those papers which are of more general interest in the current volume of the Brompton Hospital Reports include a description of the surgical treatment of twenty cases of constrictive pericarditis. The nature of the disease is con-

sidered, its differentiation from right-sided heart failure is well illustrated, and there are practical details of the management and operative technique of particular interest to the surgeon. This is a really worthwhile paper on a controversial subject.

A paper on pulmonary hydatid disease reviews the surgery of 30 cases. The clinical and pathological types are described. Operative details are considered, and, though the conclusions do not comply with others recently published, the operative results speak well for the author's technique.

An account of 4 cases of lobar collapse, which has developed bronchiectasis, adds knowledge to the mechanism of bronchial dilatation. The point that this is not an irreversible process is stressed.

Sarcoidosis is diagnosed only with reserve, but in a paper relating to 135 cases, in which this diagnosis was considered, the point is made that in only 16 cases was sarcoidosis confirmed. Some features of the disease lead the author of this article to regard the overlap with tuberculosis as most probable, and the argument is backed by some excellent radiological illustrations.

Chest surgeons will find a good argument for the use of decortication in cases of infected hæmothorax. A large series of war injuries is described and how the benefit in uninfected cases and the advent of penicillin led to the use of the operation in those cases with infection—a step which the authors prove to be fully justified.

The anatomical abnormalities of the bronchi and the lobes of the lung are grouped in a simple classification, which makes the facts more easy to absorb, so that the diagnosis of these rarities in life would seem almost easy.

For those interested in the pneumokoniosis there is a description of recent experiments done on rabbits, using asbestos fibre cut in two standard lengths. The foreign body reactions are compared and the site of the lung affection is shown to differ with the length of the fibre inhaled. The simultaneous inhalation of aluminium dust has no effect in the rabbit.

There is a description of three cases of superior vena cava obstruction due to an upper mediastinitis. The ætiology of this rare, chronic infection is discussed, but it remains obscure, and the author concludes that radical surgery is of no avail.

A case of bronchostenosis in adult pulmonary tuberculosis has a particular topical interest, and in another short article an unusual case of myelogenous leukæmia with radiological changes in the lungs, and unassociated pathology in other organs, is reviewed.

Some medico-legal aspects of tuberculosis are brought to light through the description of claims for compensation. The connection between trauma, sudden or prolonged, is discussed in this very interesting article.

The notes on the physiotherapy of chest cases, produced by the physiotherapy department of the hospital, are of very great value to anyone associated with such patients, and are worthy of publication as a separate monograph.

Bronchiogenic Carcinoma and Adenoma. By B. M. FRIED, M.D. Pp. 306. Baillière, Tindall and Cox, London, 1948. Price 33s.

This book is attractively produced and well arranged, so that a casual glance conveys the impression that it bids fair to be a classic of its kind. The chapter headings are comprehensive and logical in sequence, and each chapter is followed by an excellent and extensive bibliography; but closer acquaintance is disappointing, for the work is very uneven.

A good account of the incidence of bronchial carcinoma is followed by

a discussion of the histogenesis, which is provocative but too dogmatic to be acceptable to all pathologists. There is a simple classification, both macroscopic and microscopic, which promises to be most useful, but is spoiled because there is no detailed description of the various histological groups. There is an interesting account of the epidemic of bronchiogenic carcinoma in the Schneeburg and Jáchymov miners and its possible relation to exposure to radon emanations. The chapters on clinical manifestations are adequate, if somewhat pedestrian, but too many case-histories are given in the text, and some of the essential facts have to be gleaned from them. The occurrence of concomitant pulmonary tuberculosis in 10 per cent. of the author's 300-odd cases of bronchial carcinoma does not justify the inclusion of eleven long protocols of these patients. A really useful chapter is devoted to the description and discussion of hypertrophic pulmonary osteo-arthropathy, a subject which is much neglected in other standard works. The author advances the interesting hypothesis, supported by much clinical data, that this condition may be related to acromegaly and be due to an endocrine defect. The section on radiological diagnosis is brief and very incomplete, and many of the radiographs in the text are inadequately described. It is indeed surprising to read in such a recent monograph that " the method of tomography is new and awaits further trial " ! The chapter on treatment consists of a brief survey of some of the published results of surgical and deep X-ray therapy.

The author was one of the first to recognise bronchial adenoma as a separate entity, and he gives a concise summary of the modern views regarding the origin and treatment of this tumour, together with an accurate account of its symptomatology.

The work concludes with chapters on bronchial mesodermal tumours and mediastinal tumours; these are so brief that they will be of little value to the serious student, and, unfortunately, the same must be said of the index.

A. F. F-C.

NOTICE

TEMPORARY APPOINTMENTS AT THE "FORLANINI" SANATORIA IN ROME AND NAPLES, 1949-50

ATTENTION is drawn to a number of temporary appointments tenable by medical practitioners of British nationality at the Ospedale Sanatoriale " Forlanini " in Rome and at the Ospedale Sanatoriale " Forlanini " in Naples. Appointments of this type are offered by the Istituto Nazionale della Previdenza Sociale to doctors from other countries who wish to acquaint themselves with Italian methods of treating tuberculosis. Successful candidates will receive full board and lodging in the hospital to which they are appointed. They must pay their own return fares and supply their own pocket money. The appointments are for a period of eight months: November 1, 1949, to June 30, 1950.

Candidates should send their applications to The Controller, Education Division, The British Council, 3, Hanover Street, London, W.1. They should state their age, give a full account of their medical training and experience, and furnish the names and addresses of two references. The closing date for receipt of applications is September 15, 1949.